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Blood Carbon Monoxide and Hydrogen Cyanide Concentrations In the Fatalities of Fire and Non-Fire Associated Civil Aviation Accidents, 1991-1998

Arvind K. Chaturvedi
Dudley R. Smith
Dennis V. Canfield
Civil Aeromedical Institute
Federal Aviation Administration
Oklahoma City, Oklahoma 73125

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| 16. Abstract Postmortem blood samples submitted to the Federal Aviation Administration's Civil Aeromedical Institute (CAMI) from fatal civil aviation accident victims are analyzed for the primary toxic combustion gases carbon monoxide (CO), as carboxyhemoglobin (COHb), and hydrogen cyanide, as cyanide (CN ⁻). These analyses are performed to establish possible exposure of victims to smoke produced during in-flight/post-crash fires or to CO leaked into cabin/cockpit from faulty exhaust/heating systems. The presence of both gases in blood would suggest that the victim was alive and inhaled smoke from a fire. If only COHb is elevated, then the accident (or death) could be the result of CO contamination of the interior. Information pertaining to blood levels of the 2 gases in aviation fatalities, in relation to the associated accidents, is scattered or not available, particularly with regard to toxicity. Therefore, considering that COHb $\geq 10\%$ and CN ⁻ $\geq 0.25 \mu\text{g/mL}$ are sufficient to produce some degree of undesired physiological effects, the necessary information was extracted from the CAMI's toxicology database. Biological samples from 3857 fatalities of 2837 civil aviation accidents, occurring during 1991-1998, were received at CAMI. Out of these, 1012 accidents, encompassing 1571 (41%) fatalities, were fire associated, whereas 1820 accidents were non-fire related. The remaining 5 accidents were of unknown fire status. There were fewer fire-related fatalities and associated accidents in the category wherein COHb $\geq 10\%$ and CN ⁻ $\geq 0.25 \mu\text{g/mL}$ than that in the category wherein COHb $< 10\%$ and CN ⁻ $< 0.25 \mu\text{g/mL}$. No in-flight fire was documented in the former category, but in-flight fires were reported in 14 accidents (18 fatalities) in the latter category. No fatality under non-fire accidents was found in which the levels of both gases were determined to be at or above the stated levels. There were 15 non-fire accidents with 17 fatalities in which only COHb (10-69%) was elevated, indicating that these accidents were associated with the inhalation of CO of non-fire origin. The present study suggests that the aviation fire accidents/fatalities were fewer than the aviation non-fire accidents/fatalities, with the fact that the fire-associated fatalities had COHb and CN ⁻ at levels high enough to produce some degree of impairment/toxicity. Furthermore, the study confirms that aviation accidents related to in-flight fires and to CO-contaminated interiors are rare. | | | | | |
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BLOOD CARBON MONOXIDE AND HYDROGEN CYANIDE CONCENTRATIONS IN THE FATALITIES OF FIRE AND NON-FIRE ASSOCIATED CIVIL AVIATION ACCIDENTS, 1991-1998

INTRODUCTION

Postmortem biological samples collected at autopsy from the victims of fatal civil aircraft (air carrier and general aviation) accidents are submitted to the Federal Aviation Administration's (FAA's) Civil Aeromedical Institute (CAMI) for toxicological evaluation (Public Law, 1988). The sample submission is coordinated through the FAA's Office of Accident Investigation by the National Transportation Safety Board (NTSB), which is responsible for investigating all U.S. civilian aircraft accidents. For such evaluation, biological samples are analyzed for drugs, alcohols, and the 2 primary toxic combustion gases, carbon monoxide and hydrogen cyanide. The former gas is measured in blood as carboxyhemoglobin (COHb), while the latter in blood as cyanide (CN⁻). The blood gas analyses are performed to establish possible exposure of victims to smoke produced during in-flight/post-crash fires or to carbon monoxide leaked into the cabin/cockpit air from a faulty exhaust or heating system (*Aviation Monthly*, 1991; Chaturvedi, 1995). In the majority of cases, the submitted postmortem samples are from pilots and co-pilots. However, depending upon the nature of an accident—for example, an accident involving fire—samples from other crewmembers and passengers are sometimes submitted.

It is well established that carbon-containing materials (such as cotton and polyethylene) generate carbon monoxide and carbon dioxide upon burning, whereas nitrogen-containing materials (such as Nylons, silk, and wool) also generate hydrogen cyanide (Gad, 1990; Sanders et al., 1992; Chaturvedi, 1995; Chaturvedi & Sanders, 1996; Hartzell, 1996). Since the aircraft structure is composed of a variety of carbon- and nitrogen-containing polymeric materials, there is a strong potential for the generated smoke to be rich in carbon monoxide and hydrogen cyanide. In the absence of fire, the presence of carbon monoxide in the aircraft's interior would be indicative of the malfunctioning of heating/exhaust systems: Engine

exhaust would contain a negligible amount of hydrogen cyanide, since aviation fuel is primarily a mixture of non-nitrogen-containing hydrocarbons.

In-flight smoke and fire are rare occurrences in modern aviation, but if that happens, then the consequences are generally deadly (McKenna, 1996; Proctor, 1996; ABC News, 1998; CNN, 1998). Survivable crashes followed by conflagration do occur, primarily originating from fuel spills around the crashed aircraft. Aircraft occupants may survive the initial forces of such crashes, but they are frequently unable to escape from the fire environment because of physical injuries and/or performance impairment from smoke-induced toxicity and visual obscuration, leading to incapacitation and death. Post-crash fire has been determined to be the most important reason for pilot fatalities in commuter aircraft/air taxi crashes (Li & Baker, 1993). A study by the International Cabin Water Spray Research Management Group has concluded that there were 95 fire-related civil passenger aircraft accidents worldwide over a 26-year period, claiming approximately 2400 lives (ICWSRMG, 1993). A U.S. General Accounting Office publication further concluded that approximately 16% (32 accidents) of all U.S. transport aircraft accidents between 1985 and 1991 involved fire, and 22% (140 fatalities) of the fatalities in these accidents resulted from the effects of fire and smoke (GAO, 1993).

Although previous studies report the number of fatalities in smoke/fire-related aviation accidents, information on blood levels of the 2 combustion gases found in fire and in non-fire aviation accident fatalities is scattered or not available, particularly in relation to performance impairment, toxicity, incapacitation, and death. This type of information will be valuable to establish whether a particular victim succumbed to smoke before or after the plane crashed and to determine possible interactive toxic effects of these gases. The presence of both gases in the blood would suggest that the victim was alive and inhaled smoke from a fire. If both gases were not found in blood and the victim had physical and external burn

injuries, then it can be deduced that the cause of death was not the inhalation of smoke, but instead should be attributed to severe injuries. If only COHb is elevated, it can be inferred that the accident (or death) was because of the contamination of the aircraft interior environment with carbon monoxide. Therefore, the necessary information from the CAMI's toxicology database was extracted to determine the number and nature of fire/non-fire involved aviation accidents and associated fatalities wherein blood carbon monoxide and/or hydrogen cyanide reached concentrations sufficient to produce impairment/toxicity. Attempts were also made to obtain any available accident/case histories and pathological findings observed during autopsy.

EXPERIMENTAL

Since 1991, a database on toxicological findings of civil aircraft (air carrier and general aviation) accident fatalities (pilots, co-pilots, other crewmembers, and/or passengers) has been maintained at CAMI. This CAMI toxicology database was searched for aviation accidents that occurred during 1991-1998. The search was based on the criteria associated with fire and non-fire accidents in the presence and absence of the 2 combustion gases in blood. At or above 10% COHb and 0.25 µg CN⁻/mL blood, some degree of clinical manifestations of the poisonings of carbon monoxide and hydrogen cyanide has been reported (Gossel & Bricker, 1994; Baselt & Cravey, 1995). Accordingly, the selected criteria were: (i) total numbers of accidents and associated fatalities from which biosamples were submitted to CAMI for analysis, (ii) numbers of fire-involving accidents and related fatalities wherein COHb was ≥ 10% and CN⁻ ≥ 0.25 µg/mL, (iii) numbers of non-fire accidents and related fatalities wherein COHb was ≥ 10% and CN⁻ ≥ 0.25 µg/mL, (iv) numbers of fire-related accidents and fatalities wherein COHb was < 10% and CN⁻ < 0.25 µg/mL, and (v) numbers of non-fire accidents and related fatalities wherein COHb was < 10% and CN⁻ < 0.25 µg/mL.

Submitted blood samples were spectrophotometrically analyzed for COHb (Blanke, 1976; Canfield et al., 1999) and for CN⁻ colorimetrically using the chloramine-T/pyridine/barbituric acid reagent (Blanke, 1976; Lundquist et al., 1985; Sunshine, 1987a; Chaturvedi et al., 1995). Based on the sensitivities of the respective analytical methods, the cut-

off concentrations were established as 10% for COHb and 0.25 µg/mL for CN⁻. These analytical cutoffs are above the levels reported in normal tobacco smokers; that is, COHb levels in the range of 0.7 to 6.5% (Trinder & Harper, 1962; Ayres et al., 1969; Sunshine, 1987b) and blood CN⁻ in the range of 0.006 to 0.041 µg/mL (Baselt & Cravey, 1995). Although the submitted biological samples were also analyzed for drugs and alcohols by using the laboratory standard analytical procedures, the database search was focused on the aforementioned 5 criteria (see previous paragraph) to be within the scope of the present study; furthermore, passenger biosamples are not always analyzed for drugs and alcohols.

RESULTS

Fire and non-fire aviation fatality accidents

Biological samples from 3857 fatalities of 2837 aviation accidents that occurred during 1991-1998 were received at CAMI for toxicological evaluation (Fig. 1; Table 1). This aviation accident number was 77% of the total number of fatal aviation accidents (3684) investigated by NTSB for that period, as given in the FAA's National Aviation Safety Data Analysis Center (NASDAC) database, Washington, DC. The higher number of fatalities in relation to the number of accidents was obviously attributed to the presence of more than one occupant—pilot, co-pilot, other crewmembers, and passengers—in some of the aircraft of those accidents. Out of these, 1012 accidents encompassing 1571 (41%) fatalities were fire associated, whereas 1820 accidents (2280 fatalities) were non-fire related. The remaining 5 accidents totaling 6 fatalities were of unknown fire status.

As exhibited in Fig. 1, the majority of samples (80%) received came from pilots and co-pilots. Samples from other crewmembers and/or passengers were considerably lower (20%). However, the number of samples from passengers of fire-involved accidents was relatively higher (28%) than that of non-fire accidents (15%). These patterns were consistent with the fact that biological samples from passengers of fire-involved accidents are also frequently submitted, but only the COHb and CN⁻ analyses are performed.

Out of the total 2837 accidents (Table 1), COHb and/or CN⁻ analyses were performed on blood samples from the fatalities of only 1974 (70%) accidents (Table 2). Blood samples from the fatalities of the remaining accidents were either not suitable or not

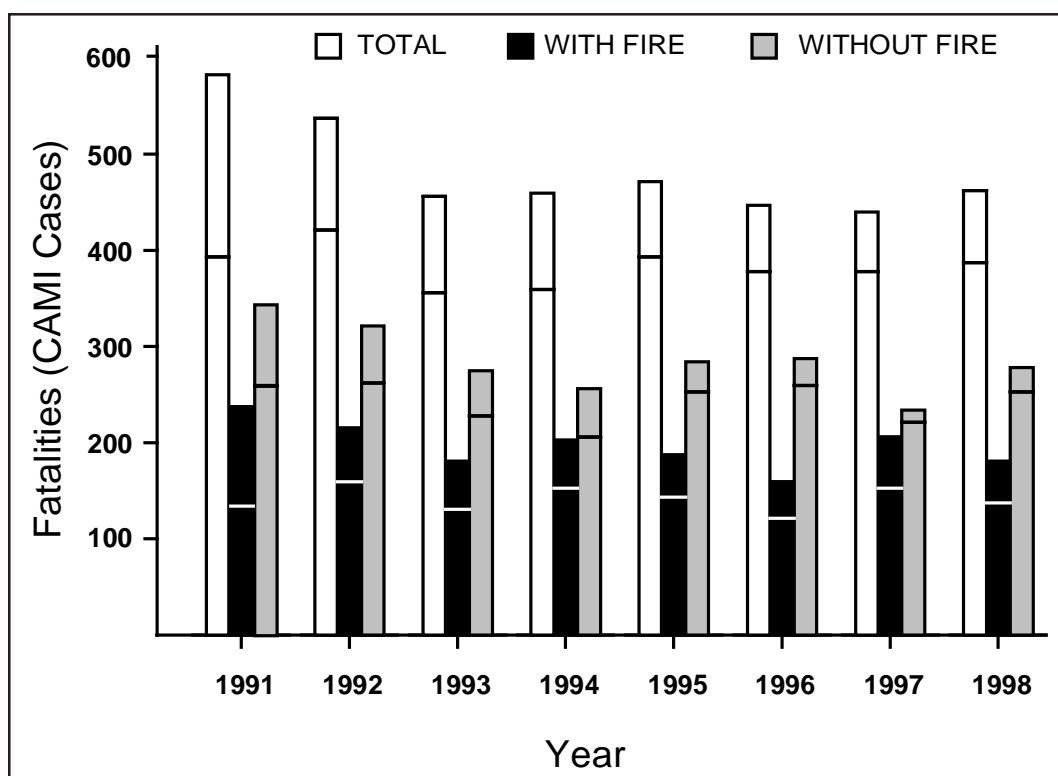


Figure 1. Number of cases analyzed by CAMI during the 1991-1998 period. Each bar has 2 parts. The lower part represents cases related to pilots (pilots and co-pilots); the upper part represents other crewmember and/or passenger cases.

Table 1. Number of aviation fatality accidents (1991-1998) from which postmortem biological samples were submitted to CAMI for toxicological evaluation.

| Year | Fatality Accidents Investigated by NTSB* | Number of Accidents (CAMI Cases) | | | Accidents With Unknown Fire Status |
|-------|--|----------------------------------|---------------------|------------------------|------------------------------------|
| | | Total Accidents | Accidents With Fire | Accidents Without Fire | |
| 1991 | 498 | 382 | 126 | 255 | 1 |
| 1992 | 512 | 397 | 146 | 251 | 0 |
| 1993 | 446 | 344 | 124 | 219 | 1 |
| 1994 | 463 | 351 | 148 | 202 | 1 |
| 1995 | 467 | 361 | 127 | 234 | 0 |
| 1996 | 423 | 337 | 102 | 235 | 0 |
| 1997 | 414 | 326 | 123 | 202 | 1 |
| 1998 | 461 | 339 | 116 | 222 | 1 |
| Total | 3684 | 2837 | 1012 | 1820 | 5 |

*Numbers obtained from the FAA National Aviation Safety Data Analysis Center (NASDAC) database, Washington, DC.

Table 2. Number of fatalities and related fire and non-fire accidents in the presence and absence of COHb and CN⁻ in postmortem blood (*Numbers in parenthesis indicate number of accidents*).

| Year | Fire Accidents | | Non-Fire Accidents | |
|-------|---|---|--|--|
| | Category A Fatalities with COHb \geq 10% and CN ⁻ \geq 0.25 $\mu\text{g/ml}$ (<i>Number of Accidents</i> *) | Category B Fatalities with COHb < 10% and CN ⁻ < 0.25 $\mu\text{g/ml}$ (<i>Number of Accidents</i> †) | Category C Fatalities with COHb \geq 10% and CN ⁻ \geq 0.25 $\mu\text{g/ml}$ (<i>Number of Accidents</i>) | Category D Fatalities with COHb < 10% and CN ⁻ < 0.25 $\mu\text{g/ml}$ (<i>Number of Accidents</i>) |
| 1991 | 17 (7) | 140 (93) | None (<i>None</i>) | 230 (194) |
| 1992 | 9 (7) | 120 (95) | None (<i>None</i>) | 220 (192) |
| 1993 | 6 (5) | 107 (83) | None (<i>None</i>) | 194 (173) |
| 1994 | 7 (5) | 103 (81) | None (<i>None</i>) | 167 (142) |
| 1995 | 11 (9) | 99 (74) | None (<i>None</i>) | 172 (156) |
| 1996 | 19 (6) | 61 (51) | None (<i>None</i>) | 179 (161) |
| 1997 | 3 (3) | 102 (77) | None (<i>None</i>) | 160 (144) |
| 1998 | 1 (1) | 74 (59) | None (<i>None</i>) | 173 (156) |
| Total | 73 (43) | 806 (613) | None (<i>None</i>) | 1495 (1318) |

* All accidents were associated with post-crash fires: No in-flight fires are reported in this category of accidents.

† Numbers represent in-flight and post-crash fire-related accidents. Relevant findings of the in-flight fire accidents are separately summarized in the Results section.

available for the analyses. Because of these limitations, blood samples from 2374 (62%) cases out of the total 3857 cases received were analyzed for the 2 gases (Table 2; Fig. 1). Only 43 (2%) of the total 1974 accidents had a total of 73 (3%) fatalities with COHb and CN⁻ levels high enough to produce some degree of toxicological effects. As shown in Table 2, the numbers of fire-related fatalities and associated accidents were lower in the category wherein COHb \geq 10% and CN⁻ \geq 0.25 $\mu\text{g/mL}$ (Category A) than the category wherein COHb < 10% and CN⁻ < 0.25 $\mu\text{g/mL}$ (Category B). No in-flight fire was documented in the former category, but in-flight fires were reported in a small number of accidents in the latter category (detailed in a later section). No fatality with the concentrations of the 2 gases at or above the stated cutoff values was found in the database under non-

fire accidents (Category C), albeit there were a considerable number of aviation accident fatalities in which these gases were not detected in blood (Category D).

COHb and CN⁻ concentrations

Although exposure to smoke generally results in the presence of carbon monoxide and hydrogen cyanide in blood, the distribution of fatalities with respect to the concentrations of each of these gases was done to evaluate the related degree of toxicity as if individually produced by each of the 2 gases. As exhibited in Table 3, the search of the database for the presence of each of the gases individually disclosed that the numbers of accidents and related fatalities with the COHb \geq 10% group (Group A) were less than the COHb < 10% group (Group B). Similarly,

Table 3. Distribution of cases in fire-involved aviation accidents with respect to the concentrations of COHb and of CN⁻ in blood (*Numbers in parenthesis indicate number of accidents*).

| Year | Fire-Involved Accidents | | | |
|-------|--|---|---|--|
| | Group A | Group B | Group C | Group D |
| | Fatalities with COHb \geq 10% (<i>Number of Accidents</i>) | Fatalities with COHb $<$ 10% (<i>Number of Accidents</i>) | Fatalities with CN ⁻ \geq 0.25 μ g/ml (<i>Number of Accidents</i>) | Fatalities with CN ⁻ $<$ 0.25 μ g/ml (<i>Number of Accidents</i>) |
| 1991 | 35 (13) | 149 (101) | 24 (14) | 175 (104) |
| 1992 | 22 (17) | 131 (103) | 14 (12) | 150 (114) |
| 1993 | 7 (6) | 115 (87) | 13 (10) | 124 (91) |
| 1994 | 12 (9) | 117 (88) | 12 (10) | 123 (97) |
| 1995 | 22 (20) | 106 (80) | 13 (11) | 136 (95) |
| 1996 | 27 (11) | 62 (52) | 20 (7) | 84 (65) |
| 1997 | 8 (6) | 107 (81) | 8 (7) | 135 (85) |
| 1998 | 9 (9) | 75 (58) | 2 (2) | 116 (71) |
| Total | 142 (91) | 862 (650) | 106 (73) | 1043 (722) |

the numbers were less in the CN⁻ \geq 0.25 μ g/mL group (Group C) than in the CN⁻ $<$ 0.25 μ g/mL group (Group D). Only about 4% of the 1974 aircraft accidents had victims with COHb or CN⁻ concentrations high enough to produce toxicological effects.

Further categorization of the fatalities based on the COHb concentration-associated toxic effects revealed that 77 of the 142 victims had COHb levels in the range of 10-20% (Fig. 2A), a level considered sufficient to produce adverse physiological effects—tightness across the forehead, headache, dilation of blood vessels, and exertional dyspnea (Gossel & Bricker, 1994). The remaining victims had COHb ranging from 21-60%, the level at which severe toxic effects such as confusion, dimness of vision, nausea, collapse, and incapacitation have been reported.

Likewise, the categorization of CN⁻ concentrations (Fig. 2B) reflects that 61 out of the 106 victims had this analyte in the range (0.25-1.00 μ g/mL) sufficient to cause mild adverse effects, and 29 victims had the analyte at the level (1.01-2.50 μ g/mL), which is considered enough to produce a moderate degree of poisoning (Gossel & Bricker, 1994; Baselt & Cravey, 1995). Sixteen victims had CN⁻ at levels ($>$ 2.50 μ g/mL) that would cause severe adverse effects, including coma and death.

In-flight fire accidents

Of the 1991-1998 accidents, 14 accidents with a total of 18 fatalities were in-flight fire related. Blood samples received from these fatalities were also analyzed for both gases. In the majority of the samples,

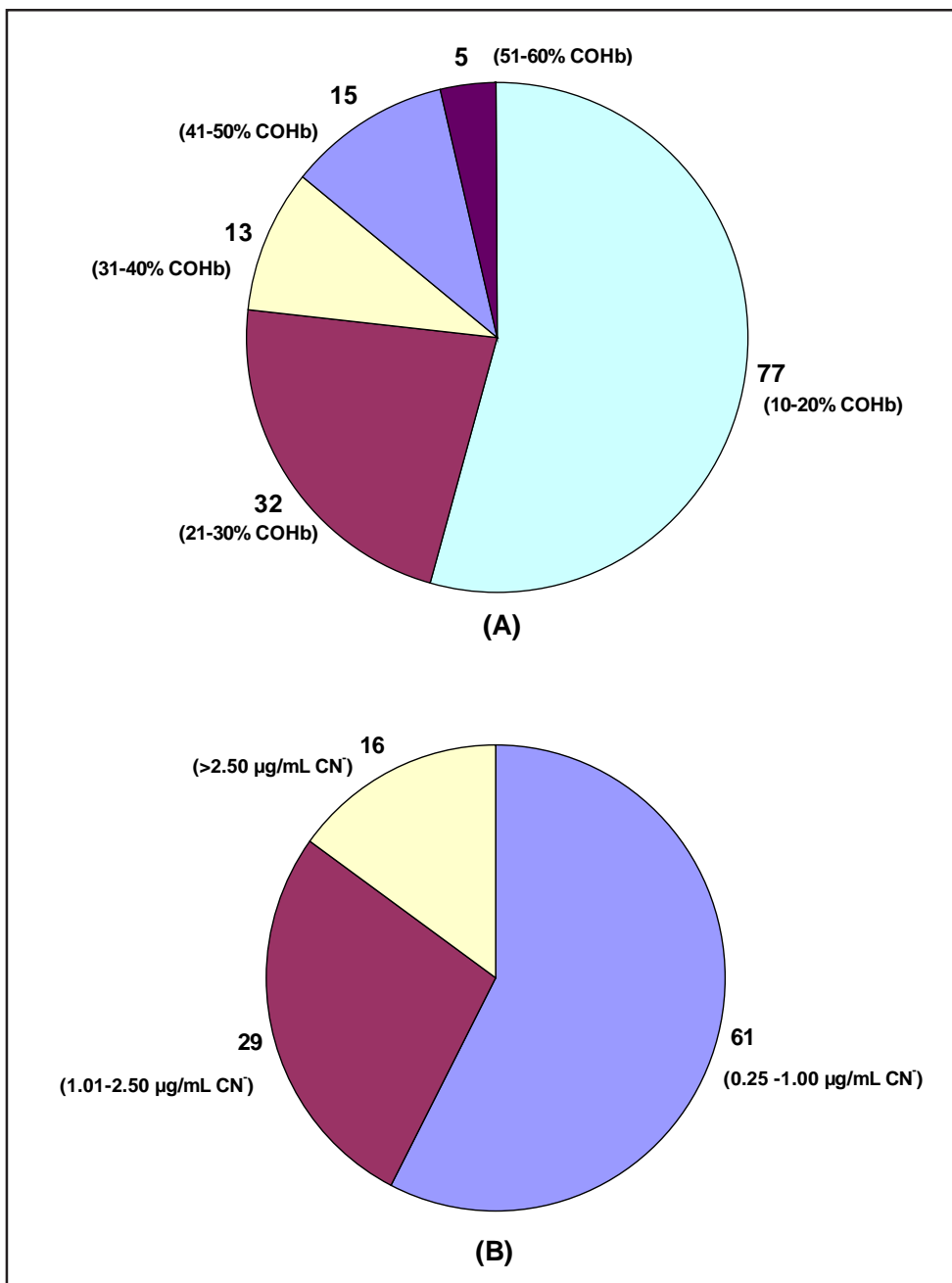


Figure 2. COHb (A) and CN⁻ (B) level-based distribution of fatalities in relation to toxicological manifestations.

COHb was not detected; the highest COHb value was found to be 5%. No CN⁻ was detected in these samples. Three samples were unsuitable for analyses. Although detailed pathological findings with all the casualties were not available, blunt trauma was prevalent in the victims. All involved aircraft were of the general aviation category, as well as homemade. The probable cause of fire with at least 3 accidents was established to be of electrical origin. The origin of fire in the remaining accidents could not be conclusively determined.

COHb in non-fire aviation accident fatalities

As compiled in Table 4, there were 15 non-fire accidents, totaling 17 fatalities (15 pilots and 2 passengers) wherein COHb was $\geq 10\%$ (10-69%). In these fatalities, CN⁻ was not detected. Based on the presence of COHb in the absence of CN⁻ and fire, it was hypothesized that these accidents were associated with the carbon monoxide inhalation during (or before) the flight. In the majority of the 15 accidents, heating/exhaust system malfunctions, pilot error, and/or carbon monoxide-induced incapacitation were

Table 4. Various investigative elements of non-fire aviation accidents that occurred during 1991-1998, wherein blood COHb levels in the associated fatalities were determined to be $\geq 10\%$.

| Year | Number of Accidents | Number of Associated Fatalities ^{*,†,‡} | COHb (%) | Aircraft Type | Probable Cause of Accident |
|------|---------------------|--|-----------------|-------------------|---|
| 1991 | 4 | 4 | 10 | Bell 206-B | In-flight structural failure |
| | | | 20 | Aronca 7-AC | Physical impairment of pilot |
| | | | 10 | Piper 28-140 | Undetermined |
| | | | 16 [§] | Gulfstream AA-5A | Physical impairment of pilot |
| 1992 | 1 | 1 | 10 [§] | Gulfstream 690-B | Severe turbulence |
| 1993 | 2 | 2 | 11 [§] | Bell 214-B | Structural failure |
| | | | 17 | Cessna 177-RG | Pilot error |
| 1994 | 1 | 1 | 14 [§] | Beech 95-B95 | Fuel starvation |
| 1995 | 2 | 2 | 12 [§] | Cessna 150-L | Pilot error |
| | | | 11 [§] | Cessna 150-M | Pilot error |
| 1996 | 1 | 1 | 41 | Mankovich Sonerai | Possible inadequate sealing of engine's firewall—carbon monoxide-induced incapacitation |
| 1997 | 3 | 4 | 43; 69 | Piper 28-236 | Faulty exhaust |
| | | | 26 [§] | Bell 206-B | Carbon monoxide-induced incapacitation |
| | | | 12 | Windex 1200 C | Pilot error |
| 1998 | 1 | 2 | 24;12 | Piper 28-160 | Suspected faulty exhaust |

*No fatality in the database was found in which COHb level was $\geq 10\%$ and blood CN⁻ was ≥ 0.25 $\mu\text{g/ml}$ in non-fire aviation accidents during the period of 1991-1998.

†In almost all of the fatalities, pathological findings were consistent with traumatic injuries.

‡Blood CN⁻ levels were determined to be < 0.25 $\mu\text{g/ml}$. However, excluded from this table were 2 fatalities associated with 2 different aircraft accidents in 1991. In their blood samples, only CN⁻ was found to be present at concentrations ≥ 0.25 $\mu\text{g/ml}$ (3.8 and 0.25 $\mu\text{g/ml}$): No COHb was detected. In the 3.8- $\mu\text{g/mL}$ CN⁻ fatality, this analyte was not detected in other tissues, nor was it found in the blood sample of another occupant of the aircraft. The selective presence of CN⁻ in such a toxic concentration in the absence of COHb could not be logically explained. It could possibly be associated with reason(s) other than smoke inhalation.

§Nicotine and/or cotinine were also detected in the blood samples.

reported to be at least contributory factors. In relation to the total number of the fatality accidents (2837; see Table 1), the percentage of the total carbon monoxide associated accidents was 0.53. Out of these, 3 accidents with 5 fatalities (12, 24, 41, 43, and 69% COHb) were determined to be attributed to the carbon monoxide-induced incapacitation/defective exhaust system.

In the 1997 accidents, the pilot with 26% COHb was reported to be a heavy cigarette smoker (1 pack a day) and postmortem examination of the body revealed chronic pulmonary emphysema. COHb was not detected in the other 3 occupants of the aircraft (data not included in Table 4). The 1991 accident pilot with 16% COHb was exposed to carbon monoxide on the ground before the flight: The pilot was a mechanic and was exposed to carbon monoxide from running engines in a non-ventilated workshop. Although the pilot's post-accident COHb level was elevated, COHb of the other occupant of the aircraft was not (data not given). Therefore, the pilot's physical impairment might have been caused by the on-ground carbon monoxide exposure.

A further search of the database revealed that nicotine and/or cotinine were also present in the blood samples of 7 fatalities, including the pilots mentioned in the previous paragraph. Pathology of all the 17 fatalities revealed that these individuals had traumatic body injuries.

DISCUSSION

Although fire-involved aviation accident fatalities could be the direct result of traumatic injuries caused by impact and/or of the inhalation of smoke generated during in-flight or post-crash fires, blood carbon monoxide and hydrogen cyanide concentrations are indicative of respiratory exposure of victims to smoke. If an accident victim was alive and inhaled smoke, then these gases should be present in the victim's system; whereas, if an individual was dead because of injuries prior to a post-crash fire, then the combustion gases should not be present in blood. With these considerations, it is inferred that the 73 victims of the 43 fire-related fatal accidents (Table 2) did not expire immediately following the impact, as both combustion gases in their blood were present in concentrations (COHb $\geq 10\%$ and CN⁻ ≥ 0.25 $\mu\text{g/mL}$) sufficient to produce some degree of adverse effects (Gossel & Bricker, 1994; Baselt & Cravey, 1995). The presence of both gases obviously suggests

that they inhaled smoke prior to death. It can be deduced that the victims in whom COHb and CN⁻ were not detected died primarily due to the traumatic injuries—the general pathological finding in the majority of the aviation accident casualties. Not surprisingly, no fatality in non-fire accidents was found in which blood COHb was $\geq 10\%$ and blood CN⁻ was ≥ 0.25 $\mu\text{g/mL}$: None of the non-fire aviation accident fatalities had both of the gases at or above these levels.

Considering fire-involved accident fatalities with COHb $\geq 10\%$ and/or CN⁻ ≥ 0.25 $\mu\text{g/mL}$, about half of them had COHb in the range of 10-20% and/or CN⁻ in the range of 0.25-1.00 $\mu\text{g/mL}$, at which concentrations both gases cause a mild degree of adverse effects (Gossel & Bricker, 1994; Baselt & Cravey, 1995). In the remaining casualties, the blood concentrations of the gases individually were high enough to produce significant toxic effects. Since carbon monoxide and hydrogen cyanide affect neurologic functions and have interactive effects (Gossel & Bricker, 1994; Baselt & Cravey, 1995; Chaturvedi et al., 1995; Hartzell, 1996), both gases, jointly, have a potential to hasten the onset of neurotoxic effects, including performance impairment or incapacitation, leading to death.

Carbon monoxide and hydrogen cyanide could be also present in blood if there were in-flight fires. However, our findings indicate that these gases were either not detected or were below the cutoff levels, suggesting no significant exposure of occupants to smoke in those in-flight fire accidents. Nevertheless, impact-associated fatal injuries were prevalent in the victims of the in-flight smoke/fire-related accidents. Although in-flight fire victims might not be exposed to significant levels of combustion gases, in-flight smoke/fire accidents have deadly consequences (McKenna, 1996; Proctor, 1996; ABC News, 1998; CNN, 1998). It is evident that there was little exposure to the toxic gases during in-flight fires. Death most probably occurred from physical injuries caused by the crash shortly after the fire started. However, the performance of pilots (and even of other aircraft occupants) in those situations could have been adversely affected by visual obscuration and ocular irritation from smoke.

In accidents associated with the carbon monoxide contamination of the cabin/cockpit, only COHb should be elevated and CN⁻ should not be present in the blood. This criterion is consistent with the findings of the present study in that no fatality was found

in which both gases were present at or above the cutoff levels in non-fire accidents. The most probable scenario for carbon monoxide poisoning to take place is the leakage of gases into the cockpit/cabin from faulty heaters and/or exhaust systems. The high COHb ($\geq 21\%$) values clearly suggest the occurrence of carbon monoxide-induced dizziness, incapacitation, coma, or death (Gossel & Bricker, 1994). Obviously, there would be relatively mild adverse physiological effects at the low COHb (10-20%) levels. At higher altitudes, there is also a probability of the coexistence of altitude-associated hypoxic hypoxia. Therefore, the effects of carbon monoxide would be further exacerbated in association with hypoxia, thereby hastening a performance impairment that may contribute to an accident. The adverse effects would be more pronounced at elevations during flight than at ground level because of the interactive effects of altitude hypoxia and carbon monoxide-induced anemic hypoxia (Allen & Allard, 1961; McFarland, 1970; Nesthus et al., 1997). A typical situation could be a pilot smoking a cigarette just before flying an aircraft or during the flight, or working in a carbon monoxide-contaminated environment prior to flying an airplane. In an actual situation, a pilot smoked a cigarette while cruising at 13,500 feet over mountainous terrain in an unpressurized aircraft and quickly lost consciousness (FAA, 1991).

The presence of nicotine and/or its metabolite (cotinine) in blood, along with the low levels (1-7%) of COHb (Trinder & Harper, 1962; Ayres et al., 1969; Sunshine, 1987b) could be an indication of tobacco smoking, but both compounds could also be present in tobacco chewers. The presence of COHb in the absence of nicotine and/or its metabolite would suggest that the elevated COHb level could be attributed to the inhalation of carbon monoxide originating from a source other than tobacco smoking. However, elevated COHb levels in the presence of the tobacco alkaloid and its metabolite would not necessarily be indicative of exposure to carbon monoxide from smoking. It could result from exposure to carbon monoxide from smoking and/or from non-smoking sources. In spite of these perspectives, the observed total number of non-fire, carbon monoxide-exposed fatalities was only 17 (0.44%) of the total number of aviation fatality cases (3857) analyzed at CAMI, suggesting that non-fire carbon monoxide exposures are rare.

Overall, the present study revealed that fire-involving aviation accidents and associated fatalities were fewer than the non-fire aviation accidents and related fatalities. The blood levels of carbon monoxide and/or cyanide found in fire-related fatalities were often considered sufficient to impair performance and/or to produce toxicity. The findings from this study further suggested that aviation accidents and fatalities associated with in-flight fires and with the contamination of cabin environment with carbon monoxide are rare occurrences.

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